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STATUS LYMPHATICUS.

THESIS

presented for the degree of M.D.

by

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PART I.

HISTORICAL OUTLINE.

## HISTORICAL OUTLINE.

Before going into the discussion of the subject, I have purposed to outline a brief sketch of the different beliefs held regarding the connection between enlarged Thymus Gland and sudden death.

In 1614 Plater called attention to the fact that enlargement of the Thymus was often found in cases of sudden death. In 1830 Kopp associated this enlargement with certain symptoms. He looked upon it as the cause of Thymic Asthma and his writings were so convincing that his belief became general. His theory was afterwards held in doubt by Friedleben in 1858, whose dictum "There is no Thymic Asthma" unsettled the former views. In 1884 Somma published a case where the cause of death was apparently due to pressure of the gland on the trachea, but it was not until Grawitz re-investigated the subject and held that pressure on the trachea was the main factor in Thymus death that this doctrine came to have its recognised place. This view received the support of Recklinghausen and others and was strongly opposed by Friedleben.

In 1889 Paltauf advocated the hypothesis of a constitutional disease, where the hyperplasia of the Thymus was a conspicuous manifestation of some general disorder, and to him we owe the name of Status Lymphaticus. He maintained that it was due to a "lymphatico-chlorotic" constitution and that the weakness/



weakness and irritability produced thereby were sufficient to account for the fatal symptoms.

Esherich developed and modified this view. He held that excessive internal secretion produced an excitability of the nervous system which rendered it sensitive to any abnormal stimulus. The condition was in fact the result of a Hyperthymisation.

These different theories as to the causation may be tabulated thus:-

- 1 Laryngospasm.
  2. Pressure on trachea and large vessels.
  3. Lymphatico-chlorotic constitution.
  4. Hyperthymisation.
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PART II.

ANATOMY OF THE THYMUS GLAND.

### ANATOMY OF THE THYMUS GLAND.

The Thymus Gland consists of two lobes which are of unequal size, but occasionally a third lobe is present. Carlyll, in Guy's Hospital Reports, describes it as made up of two symmetrical lobes but in all infants, which I have examined post-mortem, there has been an inequality in the size of the lobes.

It is pinkish in colour, of soft, fleshy consistence and surrounded by a thin fibrous capsule.

The thoracic portion of the gland is situated in the anterior and superior mediastinal spaces while there is also a prolongation upwards into the neck. Jacobi's frozen sections of the normal new-born infant show that it reaches as high as the sixth Cervical Vertebra and extends downwards to the level of the fifth Dorsal, which corresponds to the fourth Costal Cartilage in front.

From its position, it is seen that it occupies an important relation to the trachea, nerves, and great vessels of the neck. Its deep surface is in immediate contact with the pericardium, pulmonary artery, innominate artery on the right and left carotid on the left side, superior vena cava, innominate veins and trachea.

Its blood supply is free and comes from the inferior, thyroid and internal mammary arteries. In an ordinary dissection numerous vessels/



vessels can be traced going into the gland. Friedleben held that the supply was scanty - the vessels being so small and few in number that it was impossible to cause any turgescence through venous congestion. Professor Schafer, in his Histology, says "It is highly vascular ----- The lobules, the cortex especially, are abundantly supplied with capillary blood-vessels. In man the arteries penetrate to the junction of cortex and medulla and give off most of their capillaries radially into the cortical nodules".

The veins correspond to the arteries but tributaries join also the innominate veins.

The nerves are so minute as to be difficult of dissection. It is supplied by the vagus and sympathetic.

As to its development, Cunningham, in his Text Book of Anatomy, says "It takes origin as a tubular diverticulum from the dorsal part of the pharyngeal aspect of the third visceral cleft on each side. The Corpuscles of Hassall are derived from the repeated division of one cell, the products of which remain attached to each other. Formerly they were believed to be epithelial remnants but this is now known not to be the case, as the transformation of the original elements of the organ into lymphoid cells is complete".

In the new-born infant I have found the average weight of the Thymus Gland to be 2 drachms (7.7 grammes), Dudgeon has found it to be 7-10 grammes and Bovaird and/



and Nicoll 6 grammes.

The development of the gland up to the second year is not in proportion to the growth of the body, and the weight towards the end of the second year differs but very slightly from that at birth. From that age until eight years the gland remains much the same but afterwards it begins to become fibrous and to show signs of atrophy. At puberty it rapidly diminishes in size and becomes replaced by connective tissue, but Dr Alexander Bruce in 1867 showed that there always remain remnants of the gland throughout life and Waldeyer has demonstrated that degeneration is never complete.

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PART III.

FREQUENCY OF STATUS LYMPHATICUS.

ENLARGEMENT OF THE THYMUS GLAND AND SUDDEN DEATH.

The preceding description has outlined the normal condition of the gland, weighing in the new-born infants I examined on an average 2 drachms, and extending from immediately below the Thyroid Gland in the neck into the thorax to the level of the 4th costal cartilage.

That hypertrophy of the gland is associated with sudden death is undoubted, but the frequency of it is by no means great.

Richter found only one case of what he could call Status Lymphaticus out of 1797 cases of sudden death. Dr Mair's series however gives a much higher proportion. Out of 270 post-mortem examinations on children under 15 years there was enlargement in 18 cases, but in only 2 was it associated with sudden death without any other apparent cause.

I have collected the cases which occurred in the Royal Hospital for Sick Children, Edinburgh, under the care of the present Physicians, and out of 11,758 cases (children under 12 years) up to October 1911 there were 1630 deaths, 7 of which were attributed post-mortem to Acute Lymphatism. Two other deaths occurred after the removal of tonsils and adenoids in the Surgical Out-Patient Department of the Hospital.

These statistics lead one to the conclusion that the affection is not common, our findings being 1 in 233 deaths from all causes and although Mair's figures are 1 in 135 yet Richter met with only 1 out of 1797 cases/

cases of sudden death. It is undoubted that many other factors contribute to sudden death in children and that many of the recorded cases have no dependence on Thymic conditions.

The death, for instance of Professor Langerham's son, a boy of six years, a few minutes after a prophylactic injection of Behring's Anti- Diphtheritic Serum, so constantly attributed to Acute Lymphatism, may have an entirely different explanation. There are authenticated cases on record where patients have died almost immediately after a dose of horse serum, and this appears to occur apart even from what is known as anaphylaxis. In such a condition one injection of any preparation of horse serum may render the patient so susceptible to the properties of the serum that fatal symptoms may ensue on the repetition of the dose at any period after the lapse of ten days. Even, with Anti-Diphtheritic serum, a small prophylactic dose of 300 units may render a person so sensitive to the drug that should a subsequent dose of, say 5000 units, have to be given in the event of diphtheria developing at some later date, its administration might be attended with sudden death. And, thus, the death of Professor Langerham's son might have been due not to Status Lymphaticus but to a constitution peculiarly hypersensitive to horse serum.

And besides this there are many other causes of sudden death.

In Congenital Syphilis there are many such instances. Young infants succumb easily and often with/



with little warning.

Asthenic children die suddenly and frequently from no apparent cause. Post-mortem examinations reveal nothing definite microscopically.

In cases of hyperpyrexia there may occur sudden collapse without the infant looking very ill and when death is quite unexpected.

Numerous other conditions might be mentioned, such as, Retropharyngeal abscess, aspiration of food into the trachea, pressure of intra-thoracic glands and the entrance into the larynx of ascarides.

But, after all these have been accounted for, there is undoubtedly left a small residuum, explainable only by the morbid condition of the Thymus Gland. It seems to me there has been an hypertrophy of the gland, brought about by an overdevelopment of its function, and it is to this, as I shall endeavour to explain later, that death has been due. The cases which I am to describe belong to this group and the deaths were attributed to the condition known as Status Lymphaticus by the pathologists to the Royal Hospital for Sick Children, Edinburgh.

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PART IV.

RECORD OF CASES.

RECORD OF CASES.

The first of my cases is one of particular interest. She was seen at her home by her doctor who recognised the seriousness of her illness and likened it to either a hysteria or a violent chorea, but as he was not satisfied with her condition he sent her to Hospital where she was under my care as House Physician.

CASE 1.

Mary O'Hara, aged 10 years 9 months.

Parents are fairly comfortable and of moderate means.

Father is a tinsmith.

Admitted July 16th 1911 5-30 p.m.

Died July 17th 1911 1 p.m.

Complaint - Restlessness, excessive irregular movements, semiconsciousness.

Duration of illness - 4 hours

FAMILY HISTORY.

Father and Mother healthy - no history of Rheumatism or of epilepsy in the parents. Mother is very nervous and easily put about.

- 1st Child - Boy 14½ years, healthy
- 2nd " - Girl, died, aged 6 months, of Pneumonia
- 3rd " - Patient
- 4th " - Boy, died, aged 6 months, cause unknown
- 5th " - Girl, died, aged 5 weeks.
- 6th " - Girl, 5¼ years, healthy.
- 7th " - Boy, 3½ years, healthy.

There is no history of miscarriages. Mother was healthy during pregnancies.

Home/



HOME SURROUNDINGS.

House consists of three rooms, airy, not damp, but without much sunlight. Child is out a good deal in the open air and after school hours she usually goes messages.

PREVIOUS HEALTH.

Labour was natural and child was strong at birth. She was breast fed for two months and afterwards was given the bottle consisting of cow's milk, boiled and diluted with barley water. Now she has the ordinary diet of the house.

Teeth appeared at the 4th month. She walked at 1 year 4 months and spoke at 2 years.

Appetite is good but she is very subject to bilious turns. The least disturbance brings on an attack, even a drive in a tramcar being sufficient to produce it. Her mother remarks that she has to be watchful of where she allows her in case she is upset.

Her bowels are regular. She sleeps well. When six months old she had a convulsion which her father thought was due to the heat.

She had had no snuffles in infancy and no rash.

She has not been troubled with diarrhea nor vomiting, excepting during the attacks of biliousness.

There is no history of sore throat, bronchitis or any chest affection. No skin trouble such as Urticaria or Erythema circinatum.

Infectious Diseases.

Measles at 5 years.

Chickenpox/



Chickenpox at 3 years.

Whooping Cough at 2 years.

PRESENT ILLNESS.

Five days before admission she was at a Childrens' Treat and spent part of the day in wading in the sea. On Saturday, July 15th, i.e., 4 days later, she complained of headache and did not feel inclined to go her usual messages. She however went, and in the afternoon she complained of pain in the right leg underneath the knee and in the palm of the right hand and had to take to bed. She also had a feeling of stiffness in the left side of her neck. During the night she began to tremble, became restless, and slept but little. On the morning of the next day i.e. day of admission, she seemed better but remained in bed.

About 2 o'clock in the afternoon she was playing with her father's watch-chain and was quite cheery and bright. Half an hour later she was given a drink but was unable to swallow. All of a sudden she seemed to lose consciousness, threw her legs and arms wildly about and shewed no recognition of her parents.

She remained in this condition up to the time of admission, without having spoken a word since the commencement of the sudden seizure.

GENERAL INSPECTION AND PALPATION.

Nutrition is good. She is very stout and there is an excess of fat for one of her years. She is flabby and has a somewhat pasty look. The skin is acting/

acting freely. The head is of normal shape. Abdomen and legs are well developed. Thorax well formed and there is no beading of the ribs. Eyes, ears and nose show nothing abnormal. There are no glands palpable. She moans at times and gives a short cry. Respirations are irregular and rather interrupted but there are no movements of alae nasi. Pulse 120      Temperature 99.2° F. On palpation of the abdomen nothing is to be felt. There is no tenderness and no resistance. Liver and spleen are not enlarged.

#### NERVOUS SYSTEM.

She throws her arms about and they are in continual motion. At one time flexed and rigid, at another they are extended and lax. She raises each leg and then straightens it out. While one leg is being extended, the other is being flexed and this goes on continuously.

The arms are not flung about so much as the legs. They are at times kept quite rigid, simulating that of the convulsive state. If rigid, the forearm is flexed at the elbow joint and there is much resistance offered to extension. The hand is flexed at the wrist with the thumb bent in on the palm and the fingers flexed over it.

The body keeps comparatively still and she remains on her back in bed.

#### Reflexes.

Knee jerks are present and are not exaggerated.

Plantar reflex is flexor at times while at other times extensor/

extensor.

There is no ankle clonus.

Abdominal and Epigastric reflexes cannot be elicited.

Pharyngeal reflex is very sluggish.

Sensory Functions,

A pin prick is not felt over the legs, arms or body.

It is felt over the face. When it is pricked she  
t  
twinges.

Eyes are staring, pupils are equal and widely dilated  
and do not react to light. There is no squint.

#### Respiratory System.

Thorax is well formed. Respirations are irregular  
and sighing in character.

Breath sounds are vesicular with no morbid accompani-  
ments. Percussion note is resonant.

#### Circulatory System.

Apex beat is not palpable. Heart sounds are  
closed and clear.

Right margin of Deep Cardiac Dulness slightly to  
the right of the right margin of the sternum. Upper  
border is at the 3rd rib. Left border extends down-  
wards and outwards to just outside the nipple.

#### Haemopoietic System.

Spleen is not enlarged and no glands are palpable.

#### Genito Urinary System.

Urine - acid, no albumin, no sugar, no acetone.

#### Digestive System.

Lips and gums are healthy. There is a stale  
odour/



odour from the breath. Teeth are good.

Abdomen is not tender and there is no resistance to palpation. Liver is not enlarged.

#### TREATMENT AND PROGRESS.

July 16th 1911 - Admitted this evening 5-30 p.m.

Owing to her restless condition she has been put in a padded bed to prevent her injuring herself and screens have been placed around.

Urine has been taken off with a catheter.

Pupils are dilated and do not react to light.

Conjunctival reflex sluggish.

She is perspiring very freely over the face and upper part of the chest but over the rest of the body it is not noticeable to any extent.

She has been given Chloral Hydrate and Potassium Bromide aa gr  $\bar{v}$ .

Towards midnight she became quieter and slept irregularly, the movements being much less marked.

July 17th 1911 - At 7-30 a.m. she became suddenly worse. The conjunctival reflex was gone, the pupils were not so dilated and quite active to light.

Temperature  $107^{\circ}$  F. pulse very rapid.

The countenance was ashen gray. The movements had entirely ceased and she was quite still.

Heart beats were forcible and loud on auscultation but the pulse was very weak.

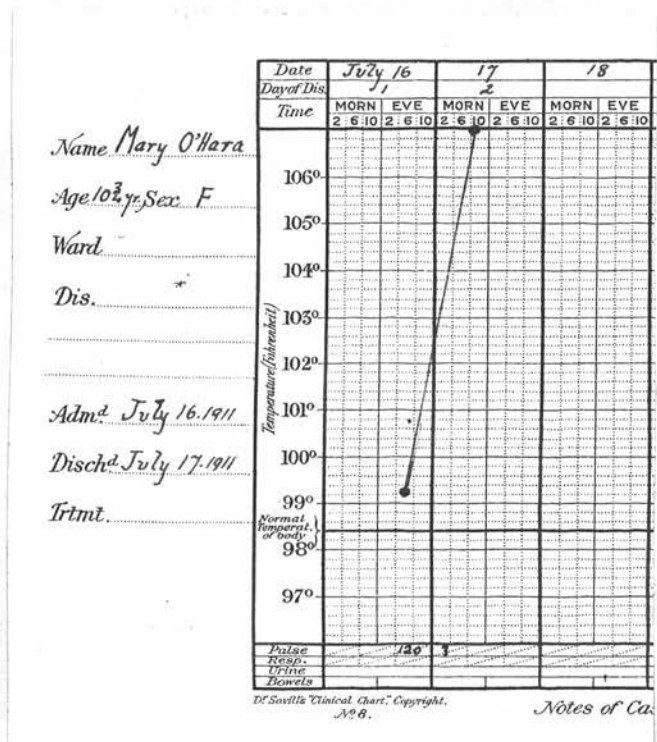
At 11-30 a.m. right border of heart was well to the right of the sternum showing marked dilatation. Six leeches/



leeches were placed over the heart.

A lumbar puncture was done. The fluid came off under ordinary pressure and was quite clear.

She gradually became worse, the ashen grey colour becoming more intense, and she died at 1 p.m.



### POST-MORTEM REPORT.

General - Well nourished, well developed child, big for her age - numerous leech bites over praecordia.

Serous Sacs - nothing of note in any.

Thymus - of very large size - thick, fleshy and spreading over the greater part of the pericardium and up to the isthmus of the Thyroid above.

Thyroid - not enlarged.

Heart - Right side moderately dilated; muscle especially of left ventricle very pale, cloudy and soft; no endocarditis, but extensive sub endothelial haemorrhages over the left side of interventricular septum.

Lungs -

Lungs - intense acute congestion throughout both, with numerous somewhat extensive haemorrhages but no definite pneumonic consolidation.

Liver - cloudy swelling.

Kidneys - cloudy swelling with congestion.

Spleen - not enlarged, pale and homogeneous looking.

Stomach and Intestine - only point of note is enlargement of solitary lymph follicles in the lower part of the ileum, standing out very prominently as hemispherical pale shot like nodules. Peyer's Patches not enlarged, nor solitary glands in large intestine or elsewhere.

Mesenteric, Bronchial and Cervical Glands - not noticeably enlarged.

Tonsils and Papillae at back of the tongue markedly enlarged - the latter coarse looking.

Tonsils are about the size of hazel nuts and in one of them is an acute abscess about the size of a small bean, not merely a dilated crypt of which there are many in both tonsils containing thin mucus.

Brain - intense general congestion but otherwise no gross lesion.

Summary. Status Lymphaticus.

I have taken sections of the different organs and the following is a description of their microscopic appearances.

Heart - muscle healthy. Nuclei distinct and well stained. Small amount of fibrous tissue interspersed in/

in places. No evidence of fatty degeneration or infiltration.

Appearance of very early cloudy swelling.

Lung. Areolar walls show much finely divided carbon pigment. They appear cellular and are thickened from dilatation of their capillaries which are full of Red Blood Corpuscles.

Kidney. Glomeruli distinct and epithelial lining intact with definite nuclei. No invasion of the space with blood cells. Slight congestion of a few capillaries near the surface and in the pyramids.

Some tubules show cloudy swelling of no marked degree.

Spleen. Pulp not very dense. Malpighian bodies very distinct with dark periphery and lighter centre. Round them is a belt of congested vessels. Malpighian bodies are collected in groups about the middle of the spleen and none near the margin. The cells of the centre of the Malpighian bodies are very indistinct with large irregular faint nuclei. Here and there near the centre are collections of brownish granules as if enclosed in a cell.

Further out is a zone of smaller dark nuclei round which are again the lighter ones.

In the pulp the cells are more mixed and the pigment is less in amount.

Trabeculae evident with blood-vessels.

Thymus - The character of the gland differs very considerably at different points. It is divided up into/

into lobules by trabeculae.

The medullary portion of each lobule is made up of loose texture with bloodvessels, large pale nucleated cells and corpuscles while the cortex is more densely packed with small darker cells.

There are many different types of cells.

At certain points especially around blood-vessels there are many eosinophil cells. They show the four different varieties described by Dr Leonard Dudgeon.

- (1) Large cells with darkly stained, granular, round nucleus eccentrically arranged. Cell body full of coarse eosinophilic granules.
  - (2) Similar cells with a non-granular nucleus.
  - (3) Cells with transitional nucleus.
  - (4) Ordinary polymorphonuclear eosinophils of the blood. Hassall's Corpuscles do not appear to be increased in number.
-



REVIEW OF CASE I.

In the review of such a case there are certain very outstanding features. In the early stages i.e. on admission, the rapidity of the pulse was very striking, and it increased, until six hours before death it was so quick and small as to be uncountable. The tension was good on the first examination, but as the case advanced, the pulse became weak with a low blood pressure while the heart beat as estimated by auscultation was quite forcible. This rapid pulse was a constant feature in all the cases and in those in which the observation was made the difference in the force of the heart beat and that of the radial pulse was also noted.

Attention was next drawn to the exophthalmos which was apparently of quite recent origin, while the pupils were widely dilated with the conjunctival reflex sluggish. The patient was not in that state of unconsciousness sufficient to account for the eye signs, and the exophthalmos could not be explained on those grounds. There was apparently something whereby the eye conditions and the Tachycardia were associated - a combination of signs such as we see in Exophthalmic Goitre, but with this essential difference, that in the latter the process is of slow beginning and of long continuance while in Status Lymphaticus it is sudden in its onset and being of the fulminating type ends rapidly. In the later stages the Exophthalmos wore/

wore off and the pupils became not so dilated as the stimulus weakened just as it did in the case of the heart.

A further interesting point was the fact that the perspiration was so profuse over the face and upper part of the chest as compared with the rest of the body, and this in itself is suggestive of local rather than central irritation. In a true toxic condition such as a septicaemia the skin acts generally and the perspiration is diffuse throughout.

Another peculiar and perhaps the most obvious sign was the continual movement. The movements were so general and so varied in type that they were apparently due to some irritation of the cerebral cortex. That there was no definite organic lesion was shewn by the Post-Mortem examination. It was unlike Chorea in that there was none of the wriggling, shrugging, grimacing of that disease. It could only be confounded with hysteria which, in its many phases, may simulate any morbid affection.

What is generally associated with Status Lymphaticus was lacking in this case. There was no suggestion of rickets in the history, and no evidence of it on clinical examination. The patient was also beyond the age when the effects of rickets are most marked, and when the weakness consequent on the acute disease is most disastrous to the child. Many of the writers describe the intimate relationship between rickets and Status Lymphaticus, but it is very probable that/



that the association of the two diseases is quite incidental, as rickets is so common a disease among infants, and it is only but likely that it should be found in a certain percentage, whatever the manner of disease they suffer from.

A remarkable fact in her history when taken in conjunction with the other cases was that of bilious turns. Any mechanical disturbance, even so slight as the shaking produced by a drive on a tramcar, was sufficient to excite an attack. It seems as if the nerves supplying the parts affected were under a constant state of irritation and the slightest over stimulus resulted in an attack of vomiting. Although there was no vomiting associated with her last illness, yet most of the other cases showed it very regularly and in some it was the leading source of complaint.

Two other cases of Status Lymphaticus were admitted to the Royal Hospital for Sick Children, Edinburgh, during my term of office as Resident Physician, and, although they were not under my care, I give in the following pages a record of them. I have taken sections of the different organs and append an account of the microscopic appearances of these.

#### CASE 11.

Helen McMorrow, aged 5 months.

Father is a labourer.

Admitted 24th April 1911.

Died 25th April 1911 1-45 a.m.

Complaint/



Complaint. Diarrhoea and Vomiting.

Duration 10 days.

FAMILY HISTORY.

Father and Mother healthy.

1st child)	}	twins - one died aged 7 weeks, the other is healthy.
2nd "		
3rd "		miscarriage at 4½ months.
4th "		Girl died, aged 12 months, of measles.
5th "		Patient.

Mother was in the Royal Infirmary, Edinburgh, while she was pregnant with the patient.

HOME SURROUNDINGS.

Windows are kept open day and night and child is taken out daily.

PREVIOUS HEALTH.

Birth was a breech presentation. Child was healthy. As mother had no milk child was bottle fed. Cream set up Diarrhoea and had to be stopped. She now gets milk and water equal parts with a little sugar. This has been stopped for the last two weeks. She has no teeth.

Bowels are constipated as a rule and she requires Castor Oil, but for the last ten days she has had diarrhoea with green slimy stools but with no curd in them.

She has had vomiting for the last two days.

She has had a slight cough of late.

There has been a discharge from the right ear for the last six weeks.

Infectious Diseases.- none.

Present/

PRESENT ILLNESS.

Since diarrhoea set in 10 days ago she has steadily lost weight and has had no appetite. On the day before admission she suddenly became much worse. She was quite strong before her illness started.

During the last two days she has cried a great deal.

GENERAL INSPECTION AND PALPATION.

Child small, emaciated and poorly developed. There are dark rings round the eyes and the eyes are sunken.

Head is well formed. She is perspiring freely over it.

No signs of rickets. Abdomen flaccid: no glands palpable; Liver and Spleen not enlarged.

Digestive System.

Lips, tongue and throat healthy. She has vomited since admission and she has had several loose stools.

Circulatory System.

Epigastric pulsation occasionally felt.

Heart sounds are clear and closed and more forcible than palpation would lead one to suspect.

Pulse rapid and weak.

Respiratory System.

Breathing is mainly abdominal.

Breath sounds are vesicular with no morbid accompaniments.

Percussion note is resonant.

Nervous System.

Fontanelle level with the vault of the cranium.

No head retraction nor spinal deformity.

Child/

Child goes into irregular movements but has had no definite convulsions.

Knee jerk cannot be elicited.

Plantar reflex extensor.

#### TREATMENT AND PROGRESS.

April 24th 1911 - Admitted in forenoon. Pulse became rapid and respirations went up to 70 per minute. She was given whisky m<sup>x</sup> 4 hourly.

April 25th 1911 - Died at 1-45 a.m.

#### POST MORTEM REPORT.

General - numerous petechial haemorrhages, cutaneous and subcutaneous, infant small, poorly developed and emaciated.

Serous Sacs - nothing of note save general and marked enlargement of mesenteric glands and numerous petechial haemorrhages over peritoneum and pleurae.

Thymus - much enlarged, covering practically the entire pericardium posteriorly and laterally with thick fleshy layer of gland.

Lingual Papillae - distinctly enlarged.

Tonsils not much enlarged and cervical glands not enlarged.

Heart - muscle pale, soft and flabby.

Lungs - nothing of note save moderate congestion at bases and petechial subpleural haemorrhages.

Liver and Kidneys shew cloudy swelling and fatty change.

Spleen - small dark Malpighian bodies not atrophied.

Stomach and Intestine - patchy catarrh most marked in lower part of small and large intestine. Peyer's Patches and solitary glands of small gut swollen.

Thyroid/



Thyroid and Suprarenal - nothing of note.

Summary. Gastric Intestinal Catarrh in a case shewing the morbid anatomy of Status Lymphaticus.

Microscopic appearances of Sections of the Organs.

Heart - no evidence of fatty degeneration, veins very full - vessels healthy.

Lung - section appears very cellular; in parts alveolar walls easily distinguishable and cavities full of catarrhal large mononuclear cells. Some alveoli plugged with fibrin.

Spleen - nothing abnormal. In places the supporting framework readily seen. Cells not so closely packed as usual.

Suprarenal - Cortical cells well defined and shew many darkly stained granules. Medulla disintegrated and appears small in amount.

Thymus - Fibrous stroma with patches of apparently lymphoid tissue in midst of this; many circular spaces with circularly arranged cells and fewer nuclei.

## REVIEW OF CASE II

This case is complicated by the presence of diarrhoea for ten days. Whether this diarrhoea was set up by an excessive irritation due to the Thymus enlargement such as is seen in Exophthalmic Goitre, where the occurrence of frequent loose stools is so common, or whether it was due to a true Gastro-Enteritis is an open question. That patches of catarrh were found in the intestine on Post-Mortem examination points rather in favour of this latter hypothesis, and it/

it is possible that the diarrhoea, which began as a separate entity, was the cause of setting up the irritation that produced the Thymus death. It is only but likely that there is an undue irritability or excitability of the heart which responds readily to any reflex stimulus and thus lights up the latent state of what is known as Status Lymphaticus.

In this case, as in Case I, we find the rapid weak pulse with a heart beat that is disproportionately strong.

The vomiting may have been due to the Gastric Catarrh, but from the fact that it occurred so late in the illness it is probable that it was related more closely with the condition of the Thymus. It was present in most of our cases.

There is, again, no history of or clinical evidence pointing to rickets, although the child may have been too young (5 months) to show any definite signs.

### CASE III.

Alexander Stobie, aged 9 months.

Father - Brassfinisher.

Admitted - 26th April 1911. Died on the same day.

Complaint - Fits for one day.

### HISTORY.

Patient was perfectly well up till day before admission, when he began to vomit whatever was given him. During the night he became feverish and about 9 o'clock on the day of admission he commenced to have fits.

He/

He became very blue and was brought to Hospital and was admitted.

#### STATE ON ADMISSION.

He was collapsed and extremely cyanosed. There was nothing definite to be made out except a few rhonchi in the right axilla.

#### TREATMENT AND PROGRESS.

On admission Temperature 100-4° F. Pulse uncountable. Respirations 52 per minute.

The heart was not very weak but the pulse was extremely feeble.

Was given Brandy, and two hot bottles were placed in his cot. He had several convulsions with only an interval of a few minutes between each.

Given Chloral Hydrate gr  $\bar{V}$  and Pot. Bromide gr  $\bar{lll}$  per rectum. He got gradually worse and died shortly after admission.

#### POST-MORTEM REPORT.

General - Very well nourished, plump, well developed child. Rigor Mortis slight.

Serous Sacs - nothing of note except numerous enlarged hyperplastic glands in mesentery. No naked eye evidence of tubercle.

Thymus - much enlarged, thick and fleshy - extending over practically the whole front and sides of the pericardium.

Thyroid - not enlarged; nothing to note.

Heart - except for cloudy swelling no obvious atrophy.

Lungs - extensive collapse of lower border of lower lobes/



lobes - some congestion but no pneumonia.

Liver and Kidneys - marked cloudy swelling and congestion.

Spleen - slightly enlarged, deeply congested and soft.

Stomach - nothing of note.

Intestine - Peyer's Patches and solitary lymph follicles stand out remarkably and extremely hypertrophied.

This hypertrophy is present throughout but is most extreme just above the ileocaecal valve.

Brain - marked congestion of meninges and grey matter - no meningitis.

Summary - Acute Lymphatism..

Microscopic appearance of sections of the organs.

Heart - Striations of Fibres distinct in places while in others more broken up. Nuclei distinct. Shows cloudy swelling of the muscle.

Liver - cells granular with well-staining nuclei - Some cells are vacuolated. In parts the cells are widely separated by dilated capillaries. Shows cloudy swelling in places.

Kidneys - show cloudy swelling.

Spleen - Centre of Malpighian bodies thinly filled - great injection of blood spaces, otherwise structure normal.

Mediastinal and Mesenteric Glands - very little fibrous tissue - circular masses of darkly stained small cells - no giant cells and no trace of Tubercle.

Intestine - Section through lymphoid nodule shows very open structure just like that of Malpighian bodies in the/

the spleen.

### REVIEW OF CASE iii

The case was unfortunately so shortly under observation that few notes had been taken. It is of interest that the peculiar discrepancy between the strength of the heart beat and that of the radial pulse is here again noted. That it should be present also in the two previous cases leads one to the conclusion that it is more than incidental.

The vomiting might be due to a reflex set up by the condition of the heart just as it is in Diphtheria, where it is known to mark the first indication of cardiac dilatation.

The other cases which I give an account of are taken from the former records of the Royal Hospital for Sick Children, Edinburgh, and I have to thank the Physicians in charge for kindly permitting me the perusal of them.

### CASE IV.

Thomas Roden, aged 7 months.

Admitted Jan 23rd 1904, 11 p.m. and died on Jan. 24th  
12-15 a.m.

Complaint - vomiting since morning.

Family History - Parents healthy and patient is the first child.

### HOME SURROUNDINGS.

Home is satisfactory but child is seldom taken out.  
Previous/

PREVIOUS HEALTH.

Labour natural and child was healthy and breast fed until 4 months after which was given condensed milk. Has no teeth.

Had an attack of Bronchitis when 4 months old and has had recurrent attacks since. Has not been subject to vomiting.

Infectious Diseases - none.

PRESENT ILLNESS.

Child appeared quite well on the night before admission and was sitting up and laughing. In the early morning he wakened up and seemed very ill. He vomited everything that was given him and lay with his legs bent up. He was given Castor Oil but as he became worse during the day he was brought to Hospital.

STATE ON ADMISSION.

Child looks moribund and extremely cyanosed. Nutrition and development good. Expression Hippocratic; conjunctival reflex gone; shows beading of the ribs, distension of the abdomen and lateral compression of the chest.

Respirations 45 per minute: Pulse imperceptible; Heart sounds just audible and running. Breath sounds vesicular with accompanying moist râles but no definite dulness. Pupils equal and slightly dilated. No squint. Swallowing is difficult. Arms and hands go into tonic contractions at times and become stiff.

Knee jerks present.

Treatment/



TREATMENT AND PROGRESS.

Admitted Jan. 23rd 1904 at 11 p.m. and died about an hour later. Was given whisky  $m\bar{v}$  twice, strychnine  $gr\frac{1}{100}$  hypodermically, and a rectal saline  $\frac{3}{4}$  which was not retained. He never rallied and died very slowly the lividity gradually increasing.

POST-MORTEM REPORT.

Body showed well marked lividity. Beading of the ribs marked. Lungs showed oedema but no Pneumonia. Heart muscle slightly pale and right side dilated. Liver and Kidneys acute Venous congestion. Spleen enlarged 3 to 4 inches and showed acute Venous congestion.

Thymus gland enlarged and also the mesenteric glands; on section no evidence of tubercle. Peyer's Patches normal; no enlarged mediastinal glands; Circumvallate papillae of tongue not enlarged; no adenoids; brain oedematous but otherwise nothing abnormal.

REVIEW OF CASE IV.

Just as in Case III, vomiting is here the most distressing symptom. It came on suddenly and from no obvious cause. The origo mali as supposed in the previous case, might have been the heart condition, and it is noted in the records that the rate was running, and on Post-Mortem examination the right heart was found to be dilated.

The facies is described as Hippocratic, but this may/

may possibly be something similar to the facies of Case I, where there was a certain degree of exophthalmos with dilated pupils.

There was also the convulsive type of movement of the arms and hands.

In this case as distinguished from some others there is strong evidence of the presence of rickets.

#### CASE V.

Charles Elliot, aged 5 months.

Admitted July 4th 1908 at 8 a.m. and died at 9-45 a.m.

Complaint - Breathlessness and cyanosis.

#### FAMILY HISTORY.

Father is a painter and healthy. Mother is subject to Asthma. No history of Tubercle.

1st child died from convulsions.

2nd " patient.

#### HOME SURROUNDINGS.

A one roomed house with sunlight and fresh air but child is not taken out much.

#### PREVIOUS HEALTH.

Bottle fed from birth on Allenbury's Food. No signs of Rickets; digestion good; no diarrhoea nor vomiting; no bronchitis nor otorrhoea; no infectious disease.

#### PRESENT ILLNESS.

Two days before admission he became very irritable and would not sleep. His mother thought he had inward/

ward pain. On the day of admission he suddenly became blue in the face. He seemed to have taken a fit and was brought to Hospital. He had perspired freely and had not slept since his illness began.

#### STATE ON ADMISSION.

Patient moribund and breathing very rapidly. Temperature 102° F. The heart sounds could not be made out definitely as the rate was so rapid and the breathing was loud.

#### TREATMENT AND PROGRESS.

Patient was given a hot mustard bath and also stimulants but died within two hours of admission.

#### POST-MORTEM REPORT.

There was a very enlarged Thymus while the glands were also enlarged.

#### REVIEW OF CASE V.

Little can be derived from the examination as the child was moribund on admission and died shortly afterwards.

The rapidity of the rate of the heart is noted and is in keeping with what has been found in the other cases.

There is no definite history of the presence of fits, but the irritability shown before admission may have been of the nature of the excessive restlessness seen in previous cases.

#### CASE VI./



CASE VI.

John Paul, aged 11 months.

Admitted May 31st 1909 at 5 a.m. and died 5-45 p.m.

Complaint - Fits.

FAMILY HISTORY.

Father is a labourer and healthy. Mother healthy.

No History of Tubercle.

1st child died of Diarrhoea aged 7 months.

2nd " patient.

HOME SURROUNDINGS.

Two-roomed house with little sunlight. Child has not been taken out much.

PREVIOUS HEALTH.

Labour difficult. Child healthy and breast fed for first three months and since then has been given milk and oat-flour in equal parts. Has two teeth. No infectious diseases.

PRESENT ILLNESS.

Patient always has had diarrhoea, but otherwise seemed well until the morning of admission when he took two convulsions within an hour of each other. He has had a chronic cough but his appetite has been good. He perspires freely and particularly about the head. Two days ago he had an attack of vomiting.

STATE ON ADMISSION.

Badly developed, poorly nourished child of very pale complexion. No cyanosis nor oedema. Skin very moist. Marked venous radicles on scalp. Fontanelle about/

about the size of a penny and depressed. Hair scanty.  
 Chest narrow - no rosary - abdomen prominent.  
 Liver and spleen not enlarged.  
 Pupils equal and do not react to light.  
 Knee jerks exaggerated.

Breath sounds vesicular with a few rhonchi; Respirations 32 per minute. Heart sounds closed - pulse 180 per minute. Temperature 103° F.

#### TREATMENT AND PROGRESS.

Child had six fits after admission and gradually sank after the last one. Temperature went up to 107° F before death.

#### POST-MORTEM REPORT.

Marked hyperplasia of lymphoid tissue throughout the body. Thymus was large and Peyer's Patches were very prominent. There were a few small ulcers in the intestine which was evidently the seat of a long standing enteritis. On the cerebellum was a small cyst about the size of a large pea which projected into the fourth ventricle; otherwise there was nothing intracranial discovered which could account for the convulsions.

Diagnosis:- probably convulsions from and associated with Lymphatism; also enteritis.

#### REVIEW OF CASE VI.

The child was apparently of a delicate constitution, and the drain of persistent diarrhoea had left him poorly nourished and more susceptible to any other/

other affection. He shared with most of the other cases a poor family record. He was the second child and the first had died of Diarrhoea at 7 months. It is a striking fact that in all the cases there had been deaths among the other children. In Case iii however the family history has not been recorded and in Cases iv and vii the patient was the first child.

There are again to be noted here the rapidity of the pulse, the presence of fits, and the undoubted evidence of the existence of rickets.

On Post-Mortem Examination, in addition to the enlargement of the Thymus there was general hyperplasia of the lymphoid tissue. This hyperplasia of lymphoid tissue in some part of the body has been shown to be constantly present in the cases described.

#### CASE VII.

Kate Turney, aged 8 years.

Admitted Jan 18th 1907 - Died Jan 19th 1907.

COMPLAINT. Swelling of face and abdomen.

#### FAMILY HISTORY.

Mother is a charwoman and is healthy.

1st child - patient.

#### HOME SURROUNDINGS.

One roomed house - windows open continually.

Child well and carefully fed.

#### PREVIOUS HEALTH.

Labour normal. Child breast fed until fifteen months/



months, but after eight months was given also milk and bread. Teeth appeared at eight months. Walked at two years. Subject to Bronchitis.

INFECTIOUS DISEASES - Measles at 1 year; Whooping Cough at 3 years.

PRESENT ILLNESS.

Two days before admission patient did not look well and she felt tired. During the night she was very restless and on the following day her face was noticed to be swelled. Her feet and legs became swollen later and she was brought to Hospital.

STATE ON ADMISSION.

Well-grown, plump child- marked puffiness of face and oedema of ankles. Chest well formed. Abdomen prominent; Liver and spleen not enlarged. Respirations 36 per minute. Breath sounds harsh vesicular with crepitations at left base posteriorly. Patient has a short hacking cough.

Apex beat outside nipple line and impulse forcible and heaving in character. Pulse rapid and of high tension.

Pupils equally dilated and contract to light.

Plantar reflex, Knee jerks and Abdominal reflexes active.

TREATMENT AND PROGRESS.

On admission patient was put in a hot bath and afterwards in a wet pack. Skin acted well.

Jan 19th - Patient vomited four times since admission./



admission. Breathing stertorous. Lips cyanosed. Pulse rapid and tension low. Albumin and casts in urine. Oxygen and stimulants given without avail and patient died at 5-45 p.m.

#### POST-MORTEM REPORT.

Shows morbid anatomy of Status Lymphaticus; heart hypertrophied and dilated. Liver and spleen markedly congested. Kidneys show the changes of acute and chronic Nephritis.

#### REVIEW OF CASE VII.

The fact that this case was complicated with an acute nephritis depreciates from the value of the observations regarding Acute Lymphatism. Whether the Kidney affection or the Lymphatism was instrumental in causing the fatal symptoms remains open, but in all conditions, in which a definite disease is discovered, one is inclined to attribute death to it rather than to the rarer and more obscure malady.

The vomiting may have been uraemic. The strong heart beat is accounted for by the hypertrophy of the heart muscle found in the Post-Mortem Examination.

The next two cases occurred after Tonsillotomy in the Surgical Out-Patient Department of the Royal Hospital for Sick Children. They appeared to be well immediately after the operation, and it was not until a few hours later that death took place.

#### CASE VIII./

CASE VIII.

David Ramsay, aged  $2\frac{1}{4}$  years.

Admitted 20th Dec. 1910 and died on the same day.

On December 13th, he developed a sore throat with feverishness. He had difficulty in swallowing and during the night his breathing was embarrassed. He was quite recovered on Dec. 18th, and on the following day Tonsillotomy was done on both sides and Adenoids scraped. He was taken home in quite good condition in the afternoon. At 6 p.m. respirations increased in rate. He vomited after even a drink of water. He became very restless and tossed about. Diarrhoea set in and at 10 a.m. Dec. 20th, he died.

POST-MORTEM REPORT.

General - well developed and well nourished child.

Heart - no naked eye lesion except very slight turbidity of muscle.

Lungs - some congestion and a few localised areas of collapse at base.

Liver and Kidneys - show no obvious lesion.

Thymus Gland - is very markedly enlarged, extending right up to the lower pole of the Thyroid in the neck and over most of the anterior surface of the pericardium in two thick broad expansions.

Mediastinal and Mesenteric Glands - also show general and considerable hyperplasia.

Stomach and Intestine - solitary glands prominent, projecting/



projecting like little hemispherical bodies.

Brain and Cord show no gross lesion.

Summary - Lymphatism - no other obvious lesion to cause death.

#### CASE IX.

Mary Bell, aged 7 years.

Died 13th Jan. 1911.

Patient had a tonsillotomy performed in the forenoon and ten hours later she suddenly took ill and died. No record was kept of the case.

#### POST-MORTEM REPORT.

Child fairly well developed and well nourished. Mesenteric glands enlarged and Peyer's Patches and the solitary glands of the intestine distinctly hypertrophied, especially in the lower half of the jejunum and in the upper part of the ileum.

Heart and lungs show no definite abnormality. The Thymus gland is very markedly enlarged extending nearly to the apex of the heart and laterally half way round the pericardium on each side. Superiorly it reaches to the lower end of the Thyroid. It is thick and fleshy and pale in colour.

Summary - Lymphatism with enlarged Thymus, cervical glands and intestinal lymphatic tissue.

#### REVIEW OF CASES VIII and IX.

That only two cases of Thymus death occurred in the/

the last many years in the Surgical Out-Patient Department of the Hospital, where numerous Tonsillotomies are done regularly, is almost in itself sufficient proof that enlarged tonsils and adenoids are no indication of an enlarged Thymus. Enlargement of the circumvallate papillae at the root of the tongue is common in Acute Lymphatism but yet I have seen as definite enlargement post-mortem in cases with a normally sized Thymus gland.

In all probability, the operation and the anaesthetic were the exciting agents in irritating the nerves already overstimulated by the enlargement of the Thymus.

In Case IX unfortunately no history of the illness was obtained but in Case VIII vomiting and restlessness were apparently the leading features.

I have given in detail those records as the varied histories open up a wider scope for investigation, although the cases had not all come under my own observation.

Mayo, in his article on the Surgery of the Thymus Gland, says "The condition Status Lymphaticus as a cause of death is not to be denied, yet such cases are extremely rare, consequently it is important that, for a better knowledge of the subject, those using the term as applied to sudden death should give the history in/

in detail, all records of the case, likewise the findings at autopsy, in order that the profession may obtain all possible information on this question."

I add an analysis of the cases and also a compendium, where the different signs and symptoms manifested in each are grouped together, in the hope that from such a compilation a better proportion may be arrived at.

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## ANALYSIS

	Case <u>i</u>	Case <u>ii</u>	Case <u>iii</u>	Case <u>iv</u>
Age	10 $\frac{3}{4}$ years	5 months	9 months	7 months
Complaint	Excessive Restlessness	Diarrhoea & Vomiting	Fits	Vomiting
Family History	F. & M. healthy 7 children & 3 dead	F. & M. healthy 4 children 2 dead & 1 mis- carriage	F. healthy M. ?	F. & M. healthy
No. of Patient & Sex	3rd. F.	4th. F.	? M.	1st. M.
Home Surroundings	House, 3 rooms, airy.	Windows open child out daily	?	Fair, seldom out.
Bronchitis	None	slight cough	?	since 4 months old.
Asthma	None	None	?	None
Laryngismus	None	None	?	None
Vomiting	Bilious turns	For last 2 days	Present	Excessive
Diarrhoea	None	For last 10 days	None	slight after Castor Oil
Rickets	None	None	None	Present
Nutrition	Good	Emaciated	Well nour- ished	?
Cyanosis ) or ) Ashen grey )	Ashen grey	?	Cyanosed	Cyanosis ex- treme.
Convulsions	None	None	Numerous	None
Movements	Excessive in earlier stage	Occasional & irregular		Arms stiff at times
Eyeballs	Protruding	?	?	Staring
Pupils	Marked dila- tation & no reaction to light in early stage.	?	?	Dilated
Perspiration	Face & upper part of body	Freely about the head	?	?
Pulse	Rapid 120. latterly ?	128	Uncount- able	Imperceptible very rapid
Temperature	99.2° - 107° F.	98° - 99° F.	100-4° F.	?
Knee Jerk	Normal	Absent	?	Present
Lymphoid Tissue	Solitary Lym- ph follicles enlarged	Mesenteric glands & Pey- er's patches enlarged	Mesenteric Glands, Pey- er's patch- es & solit- ary glands enlarged.	Mesenteric Glands enlarg



Case <u>V</u>	Case <u>VI</u>	Case <u>VII</u>	Case <u>VIII</u>	Case <u>IX</u>
5 months	11 months	8 years	2½ years	7 years
Breathless- ness	Fits	Swelling of face & legs	Restlessness & vomiting	?
F. healthy M. has Asthma 2 children 1 dead	F. & M. healthy 2 children 1 dead	M. healthy F. ?	?	?
2nd. M.	2nd. M.	1st. F.	? M.	? F.
House 1 room, not out much	Not out much	House 1 room window open	?	?
None	Chronic cough	Since going to school	?	?
None	?	None	?	?
None	?	None	?	?
None	2 days pre- viously	Present	After opera- tion	?
None	Chronic	None	Present	?
None	Present	None	None	None
?	Poor	Good	Well nour- ished	Well nour- ished
Cyanosis	Pale	Lips cyanosed	?	?
None	Numerous	None	None	?
Irritable		Restless	very rest- less	?
?	Sunken	?	?	?
?	No reaction to light	Dilated con- tract to light	?	?
Free	Free	Free	?	?
Very rapid	140-156	144	?	?
102° F.	100°-107° F.	98°-99.8° F.	?	?
?	Exaggerated	Active	?	?
All glands enlarged	Hyperplasia throughout	?	Mesenteric glands Peyer's patches en- larged.	Mesenteric glands Peyer's patches & sol- itary gland en- larged.

COMPENDIUM OF CASES.



COMPENDIUM OF CASES.

FAMILY HISTORY - Of the 9 cases the Family History of 6 has been recorded. Of these, 2 happened to be the first children, while there had been deaths in each of the other four families, either from convulsions, diarrhoea or unknown causes. There appears to be evidence of weakly constitutions in the children of these families which lessened their powers of resistance.

SEX - This has no determining influence. Five were males and four were females.

BRONCHITIS - Out of the 7 cases, where mention was made of it, two (Cases IV and VII) suffered from Bronchitis and one, (Case VI) had a Chronic Cough.

ASTHMA OR LARYNGISMUS - There was no mention of either in any of the cases.

VOMITING - Of 8 cases all except one (Case V) suffered from vomiting. Two cases were complicated (Cases II and VII), the first with diarrhoea for ten days and the second with acute nephritis, so that in these, the true cause of the vomiting is more obscure.

DIARRHOEA - One case was complicated with Enteritis (Case II) and one had Chronic Diarrhoea Case (VI). Only one case had diarrhoea accompanying the vomiting as a separate and definite symptom (Case VIII).

RICKETS - Out of 9 cases only 2 pointed to definite signs/ .

signs of Rickets (Cases IV and VI)

NUTRITION - Patients were well nourished and well developed in 5 out of 7 cases where the condition is recorded. The 2 other cases (II and VI) were complicated with prolonged Diarrhoea, sufficient to account for their emaciation.

CYANOSIS. - Of 6 cases 4 showed signs of cyanosis to a greater or less degree. In these cases it deepened towards death.

RESTLESSNESS AND CONVULSIONS - Of 8 cases 6 were restless and showed excessive movements; 2 (Cases III and VI) had convulsions epileptiform in character.

PUPILS - In the 3 cases where mention is made of the state of the pupils they were dilated, but as they may vary at different stages of the illness one single record is of little value.

PULSE - In the 7 cases where it is recorded the pulse was exceedingly rapid and very feeble. It varied from 120 to 156 per minute while in others it was uncountable and hardly perceptible.

TEMPERATURE - In the 6 cases temperature rose in the latter stages of the illness and in 2 it reached 107° F. (Cases I and VI)

KNEE JERKS. - Of 5 cases mentioned, in 4 it was normal or active while in 1 (Case II) it was absent. This latter/

latter case was, however, complicated with diarrhoea for 10 days and the child was weakly.

LYMPHOID TISSUE - Of 8 cases all showed definite hyperplasia of certain lymphoid tissue. The mesenteric glands, Peyer's Patches and the solitary glands of the intestine were chiefly affected.

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PART V.

DISCUSSION OF VARIOUS THEORIES.

## DISCUSSION OF VARIOUS THEORIES.

From the collection of material thus compiled and from the help of personal observation I have now gathered data on which a discussion of the various hypotheses may be based and a possible conclusion arrived at.

The leading theories that have been given the most support and which at certain times were regarded as supplying the most probable explanations are

- i Laryngospasm.
- ii Pressure on the Trachea and large vessels.
- iii Lymphatico-chlorotic constitution.
- iv Hyperthymisation.

### i. LARYNGOSPASM.

The existence of a Thymic Asthma, brought about by an enlargement of the gland setting up a spasm of the glottis, was never seriously credited in this country although the Germans were firmly convinced of its presence.

Friedleben unrooted the old beliefs in his monograph and emphatically declared "There is no Thymic Asthma". It was confused with laryngospasm such as is seen in laryngismus, but Jacobi found enlargement in only one out of a dozen fatal cases of laryngismus he examined Post-Mortem, and Friedleben out of a series of seventy-five found Thymus enlarged in only seven.

It is undoubtedly proved that laryngismus is a nervous affection often present when the nervous system/

system is undermined by rickets, and it has no connection whatsoever with what is known as Thymus death. In our series of cases there has been no suggestion of any suffocative seizures simulating laryngismus or of anything that might be confused with asthmatic attacks. The absence of any such history in a number of cases, definitely known to have hypertrophy of the gland, is sufficient evidence in itself to throw out of count the hypothesis as to the existence of a Thymic Asthma.

#### 11 PRESSURE ON THE TRACHEA.

The claims to this theory were first established by Grawitz. Jacobi gave it his support. In his "Contribution to the Anatomy and Pathology of the Thymus Gland" he says, "Now, the distance, in the skeleton of an infant of eight months, between the manubrium sterni and the vertebral column amounts to 2 centimeters. Thus it is clear that a thymus gland of the size reported above, particularly in cases of temporary congestion and swelling, is sufficient to fill the whole space, compress the neighbouring organs and result in death. Thus it is quite possible to explain an occasional case of sudden death, and the so called thymic asthma, by an hypertrophy of the thymus gland, although the large majority cannot be referred to such a cause".

In contradistinction to this view Friedleben asserts that the thymus, however much enlarged, has no effect/



effect in obstructing circulation or respiration or in affecting the respiratory nerves and it cannot be said to "disturb cerebral circulation or the innervation of the muscles controlling the glottis or be subject to a periodic turgescence by impeding circulation".

Such conflicting opinions by two eminent authorities leave the subject a very debatable one, but if one adopts the most rational and easy explanation - that of mere mechanical pressure - one is forced to the conclusion that pressure on the trachea and surrounding structures may at least help in causing death, if it be not the chief factor, and that Jacobi's argument has much to commend it.

In such cases where pressure on the trachea became marked the different signs of suffocation would be expected. In Case V. breathlessness and cyanosis were especially noticeable, but in no one case was there definite evidence of death by suffocation. Cyanosis was remarked on in 4 out of 7 cases, but this alone is no evidence in favour of pressure symptoms, as a dilated heart which is beginning to fail may give rise to it in an extreme degree. But, although pressure may not be the actual cause of death, it may go far to exaggerate the condition and more speedily effect a fatal termination.

There are well authenticated cases on record where the dyspnoea and discomfort that resulted from the pressure of the gland, were immediately relieved when the pressure was removed by operation.

Siegel/

Siegel reports the case of a boy of  $2\frac{1}{2}$  years who suffered from dyspnoea for four or five weeks. There was marked stridor, and during the acute exacerbation the cyanosis became extreme amounting to asphyxia. Tracheotomy was done without success, but a long laryngeal tube inserted as far as the bifurcation of the trachea gave immediate relief. An operation exposed the upper portion of an enlarged Thymus. It was drawn up and stitched to the fascia over the sternum with a good and lasting result, there being no further return of the dyspnoea.

Koenig took out a portion from a child aged 2 months, who had dyspnoea from the eighth day, with perfect success.

Perrucker took out the entire gland in a child aged  $2\frac{1}{2}$  years, who had stridor from the fifteenth day after birth. He had aphonia and dyspnoea. He recovered, and eight months later was perfectly well, and showed no symptoms from the loss of the gland.

In those cases of progressive growth one would have expected a slow and insidious onset, but in Acute Lymphatism the symptoms come on with startling rapidity. This acuteness of the disease may be explained on the assumption that there is a sudden congestive swelling or a sudden increase in size dependant on its own secretion. Friedleben denies the possibility of a rapid turgescence due to a venous congestion, owing to the poor supply of blood to the gland, as the vessels/

vessels are small and few in number.

Beneke offers another explanation. He suggests that the falling back of the head in children, whose muscles are so weak as to prevent their recovering their position, may have the effect of flattening the trachea against the enlarged thymus, and thus causing the sudden fatal symptoms.

He had two cases where there was flattening of the trachea post-mortem, but Paltauf has shown that this flattening can be produced artificially after death, and he says, that it is impossible to demonstrate it unless the upper part of the trachea is dissected out with the Thymus and sternum still in situ, and the interior of the trachea examined from above.

Strong criticism can be brought against these assumptions. The falling back of the head through the weakness of the muscles is an explanation applicable only to very young children. In only three of our cases was the age below nine months, and above that age such weakness would not be readily expected. This sudden pressure on the trachea would cause rapid death with all the signs of suffocation, but there is no evidence of such in our nine cases.

The other hypothesis - that of the gland undergoing turgescence from a sudden engorgement of blood or retention of its own secretion - is rather fanciful. One wonders what would cause this rapid congestion and to what extent the gland would swell.

In the post-mortem examinations there was no evidence/



evidence of congestion of the gland.

It may thus be safely assumed that pressure is rarely, if ever, the actual cause of death, but it is only but likely that the hypertrophy of the Thymus gland in preventing free play of the surrounding structures may be a possible factor.

### ... III LYMPHATICO-CHLOROTIC CONSTITUTION.

In 1889 Arnold Paltauf expostulated an entirely new theory. He looked upon the enlargement of the Thymus as only a local manifestation of a constitutional disease, which he called Status Lymphaticus. He noticed that there was a general hypertrophy of the lymphoid elements, as seen by definite enlargement of the spleen, mesenteric glands, Peyer's Patches, solitary glands of the intestine, bronchial and pharyngeal glands and the circumvallate papillae.

Added to this, there was the pale and pasty look of the anaemic, and common with what is so constant in Chlorotics, there was the increased adiposity - the cheap tissue which thrives best with impoverished blood, and increases at the cost of the more expensive fluids of the body. A further resemblance was found in the hypoplasia of the heart and aorta and blood-vessels. This was first claimed by Virchow in 1872 to be the fundamental pathological condition of chlorosis. It is said to occur frequently in Status Lymphaticus, and Carlyll says "Sufficient evidence has been reviewed to show that hypoplasia of the heart and arteries, which is /

is a prominent anatomical feature in the Status Lymphaticus, is often of itself an evidence of the congenital defect in physical development and indicates a diminished vital resistance in the organism".

It was on these grounds that Paltauf based what he called "the lymphatico-chlorotic constitution", and he considered it was those belonging to this diathesis that were the subjects of Status Lymphaticus. He held that the sudden death was due to the general weakness and subsequent cardiac paralysis.

If such were the case we should naturally expect a flabby thin walled heart shewing fatty degeneration, but in the post-mortem reports in our series of cases, where there is a record of the state of the heart muscle, there is no evidence of this condition. In analagous constitutional diseases such as Leukaemia the friable heart wall thin and soft, likened sometimes to the consistence of blotting paper, is constantly seen in post-mortem examination but in such cases there is the history of a slow decline.

In Status Lymphaticus, where as Paltauf asserts, death is due to general weakness we should expect a similar condition of the heart, but how then is it that the subjects of it are thought by their parents to be strong and healthy and in full vigour are felled by this dread disease and die?

From clinical evidence this hypothesis again fails. In Cases I, II and III it was specially noted that the heart/

heart beat was disproportionately strong as compared with that of the radial pulse. In all there was rapidity of the heart's action. How can this be explained on the ground of Paltauf's theory? The rapidity may be said to be due to the general weakness, but if cardiac paralysis subsequently supervene, we should look for a slowing heart that ceases to beat in diastole.

But what reasons can be brought forward to account for the discrepancy between the force of the heart beat and that of the radial pulse, if one accepts the hypothesis of death being due to the weakness of a delicate constitution? I think there are none.

#### IV HYPERTHYMISATION.

Escherich modified Paltauf's view and advanced the hypothesis that hypersecretion of the Thymus Gland was the real cause of the disease. He held that the excessive amount of secretion entering the blood caused an irritability of the nervous system, and the nerve centres, being left thus more excitable, responding easily to the slightest stimulus. This is in keeping with the modern idea of internal secretions. It is held that it is similar in function to the other ductless glands and that its derangement caused Status Lymphaticus just as derangement of the Thyroid may cause Myxoedema when there is a deficiency of secretion/



secretion.

To test the effect of excessive Thymus secretion Svehla made a series of experiments on dogs with the dried and watery extracts of Thymus taken from bullocks, pigs, dogs and man. The effect on the circulation was particularly noted. The extract was injected into the femoral vein and the effects on the circulation watched by the Kymograph. The conclusions he arrived at were that there was a fall of blood pressure due to weakening or paralysis of the vaso constrictors, and also an increase in the pulse rate due to direct influence on the heart. The administration of large doses was followed by excitement; dyspnoea, collapse, and death, and on post-mortem examination there were all the signs of asphyxia. These symptoms are very similar to those seen in Acute Lymphatism, and the practical bearing of the observations is that Thymus death may be caused by hypersecretion of the gland.

Swale Vincent, however, showed later that injection of any tissue, glandular, muscular or nervous, when administered in the same way, produced much the same result.

These latter experiments detract from the value of Svehla's observations, and they make it apparent that extracts of mostly all the tissues of the body when injected directly into the blood stream have a toxic action on the system.

If Status Lymphaticus were due to this excessive output of Thymus secretion into the blood stream with the/

the gland enlarged or overfunctionating, we should expect minor attacks simulating in some degree the fatal one owing to an overdischarge of secretion. But such a History is never got. The child has been well previously, and there had been nothing to warn the parents of his having "so slender a hold on life".

Again, if the Thymus plays so important a part in the metabolism of the body, and its internal secretion has so governing an influence on the health of the child, it is strange that such an organ should only be temporary and that after the first few years of life it should show signs of degeneration, and subsequently atrophy. The question may well be asked "How is the body to thrive without such a gland or what organs take on its function".

Mendel stated that there is a close connection between the Thymus and the proper development and growth of bone, and he attributed the symptoms of rickets to a disorder of the function of the gland. On these grounds he treats his cases of rickets with Thymus Gland. If such a view were correct, those subject to Status Lymphaticus, where there is hypertrophy of the gland and according to Escherich hypersecretion, should be particularly free from all signs of rickets, but this is known not to be so, and many authorities even assert that it is chiefly met with in rickety children. I cannot however entirely agree with this latter statement for out of the nine cases recorded/

recorded there were not more than two definitely rickety. It is highly probable that the presence of rickets is altogether incidental. So many children in the large towns suffer from it that, in whatever disease chosen, the proportion showing signs of rickets is high.

Perrucker removed the entire gland in a boy aged  $2\frac{1}{2}$  years and eight months later there were no signs of any deficiency arising from the lack of it. Olivier reports forty Thymectomies with no untoward result arising from the want of secretion. From these cases it is clear that the internal secretion, in so far as it governs the metabolism of the body, is far too highly overestimated.

That the Thymus has any special and governing internal secretion does not get support from experimental or clinical observations. It was considered to enlarge in cases of exophthalmic goitre and that its function was opposed to that of the Thyroid. But this enlargement of the Thymus is by no means constant - Mayo in his paper "The Surgery of the Thymus Gland" says "A persistent Thymus throughout life is a common condition ,..... It is also claimed that an enlarged Thymus may be found in all post-operative fatal cases of Grave's Disease and, consequently, the gland is always enlarged in this condition. In some of our patients who died from Grave's Disease both with and without operation the size of the Thymus certainly was not a factor in causing death, since, while persistent in many cases, it was reduced to a vestige in others".

On/



On the hypothesis that the Thymus enlarged in Grave's Disease to counteract the overfunctionating Thyroid Dr Hector Mackenzie treated twenty cases with Thymus gland, but was unable to confirm the idea that Thymus gland had any specific action in this disease. His conclusions were that its administrations had no effect on the action of the heart or the size of the goitre or on the exophthalmos.

Were the theory of hyperthymisation to be accepted, we should look for the general results of a toxæmia. The clinical evidence does not support this view. Let us find an analogy in an Acute Pneumonia. The severity of the toxins may be measured by the state of the knee jerks. If the case is a dangerous one and the patient is felled with the consequent toxæmia the knee jerks are much diminished or absent. In five of our cases the condition of the knee jerks is described. In only one of these were they absent (Case II) and in this case the child was already collapsed with a diarrhoea persisting for ten days. In other two (Cases I and IV) they are recorded as being present and normal while in Cases VI and VII they were even exaggerated. From this we infer that there is no acute toxæmia and that the hypersecretion of the Thymus gland has not the poisonous action on the nervous system, which Escherich specially claimed for it.

Again, in the examination of the urine of Case I an interesting and important point was brought out. There/

There was no reaction on testing for Acetone. It is now considered that acetone is an index of a toxaemia. Le Lorier says that in Hyperemesis gravidarum the intensity of the reaction corresponds very closely with the gravity of the patient's condition. In Eclampsia, also, it is commonly, if not always, present. These are two diseases definitely known to be due to a toxaemia, and the constant presence of acetone in both is very suggestive.

In Case I there was no acetone in the urine and this too speaks rather against the possibility of a toxaemia as being the underlying factor in Thymus death.

The Post-Mortem findings in the cases described showed little beyond the ordinary changes seen in mostly all post-mortems. There was cloudy swelling so generally present, but there was no advanced disintegration of tissue and fatty degeneration which are the typical changes of a toxic condition.

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PART VI.

POSSIBLE EXPLANATION OF THE CONDITION KNOWN AS STATUS

LYMPHATICUS AND OF THE CAUSE OF THE SUDDEN DEATH.



POSSIBLE EXPLANATION OF THE CONDITION KNOWN AS STATUS LYMPHATICUS AND OF THE CAUSE OF THE SUDDEN DEATH.

In nearly all post-mortem examinations on cases which have died with Thymus enlargement there has been found in addition hyperplasia of the lymphoid tissue in the body, and in some a certain set of glands such as, for example, the mesenteric glands show more marked hypertrophy than the others. In all our post-mortem examinations this was a constant feature and microscopic examinations shewed no abnormality in structure, beyond a definite increase in the lymphoid elements. The question naturally arises "What is the cause of this enlargement of the Thymus and the lymphoid tissue generally".

Dr Beard has done extensive research on the function of the Thymus Gland. His investigations were made on one vertebrate animal - the fish *Raja batis* - the smooth skate. He says "The plan or organisation of the skate is practically the same as that of man. No organ is found in man which has not in some part or other its counterpart in the skate and the functions of the organs of man are to all intents and purposes the same as those of like organs in the skate. With the single exception that Hassall's Corpuscles have never yet been found in the skate, though not for want of the search for them, the place of origin, the mode of development and the minute anatomy of the thymus in man essentially agree with those of the same organ in the skate".

He/

He traced the emigration of leucocytes from the thymus into the mesoblast around and further into the blood-stream, and these were found before any other lymphoid organ had developed. "At the period when leucocytes first appeared the spleen had no existence, there was no rectal gland or caecum and in fact, lymphoid structures of any and every sort were entirely wanting".

He further says "It is Kolliker's great service to have shown that leucocytes arise in the thymus from its original epithelial cells; to Gulland's researches we owe the result that the leucocytes are found in the mesoblast in the neighbourhood of the thymus; and, finally, it has fallen to my lot to shew that the first leucocytes arise in the thymus from its epithelial cells and that thus it is the parent source of the leucocytes of the body".

His investigations also show that all the lymphoid tissue is developed from the leucocytes, and that thus the lymphoid element of the body is a progeny of the Thymus gland. A few years after birth when the transformation of the original elements of the organ into lymphoid cells is complete, the Thymus begins to atrophy and its work is taken up by its offspring, the lymphoid tissue.

Dr Beard has numerous microscopic sections to illustrate his views, and with so striking evidence one is convinced of the truth of his observations.

I have applied this to the histological changes found/

found in Acute Lymphatism. I think it possible that, in such a condition, the Thymus has been overactive in its function and to meet this demand has hypertrophied. The lymphoid elements have responded to the activities of the parent source and shew a definite enlargement. In this way we have a link between the Thymus and the lymphoid tissue, and the constant hyperplasia of the latter in cases of Thymus enlargement can be satisfactorily explained.

Another striking point is the nutrition of those who are the subjects of Status Lymphaticus. They generally seem well nourished, are fat and flabby with a rather pale or sallow complexion.

It is possible that there may be an intimate relation between the adipose and lymphoid tissue, and that the excess of the adipose tissue depends on the output of the lymphoid. It is certainly probable that there is some interchange as one of the constant features in marasmus of infants is the atrophy of the Thymus. Stokes, Ruhräh and Rohrer in a paper on the "Relations between the Thymus Gland and Marasmus" drew up the following conclusions.

i Atrophy of the Thymus gland is always found in cases of Infantile Atrophy.

ii The condition of the Thymus gland is an index of the general nutrition of infants.

iii The state of the nutrition of infants may be eliminated by a microscopic examination of the Thymus at autopsy.

This/



This association between nutrition and the Thymus gland is exceedingly striking and the relationship is undoubtedly very constant.

The presence of this developmental anomaly in the Thymus and the consequently "lymphatic constitution" does not necessitate its being the factor in the production of the fatal symptoms.

The sudden death may be due to irritation set up by the enlarged thymus and yet be a separate entity quite removed from the general constitutional change. It is not known definitely if any real disturbance arises from the hyperplasia of the lymphoid tissue as Paltauf suggests and, allowing that it does, death may be due to something entirely apart.

A study of the cases, with the assistance of personal observations made on Case I during the period in which it was in Hospital, has led me to different conclusions than those I have already explained as being the leading theories regarding the causation of the disease.

To give a comprehensive explanation of the causation, as I believe it, I shall first describe the anatomy of the gland in so far as it has a bearing on the subject. The description I give is based on many dissections I have carried out in new-born infants.

On removing the sternum and costal cartilages one meets the red fleshy mass of the Thymus gland. It is more or less triangular in shape, its apex extending into/

into the neck while the base is spread over the pericardium hiding from view the greater part of the heart. A longitudinal fissure divides it almost completely into two asymmetrical lobes. A thin capsule encloses the gland, and on raising the lower part it can be easily dissected off the pericardium. The differentiation between the thick fibrous pericardium and the thin delicate capsule of the Thymus is distinct, the two being separated by fine loose areolar tissue. No nerves or blood-vessels enter the gland in this large fleshy part. As the gland is being dissected off the arch of the aorta veins are seen passing out to the innominate and higher up into the Inferior Thyroid vein.

Minute nerve twigs can be traced backwards and seem to enter the cardiac plexus. The plexus is supplied by branches from the vagus and by the upper, middle and lower Cervical ganglia of the Sympathetic. It is thus likely that it receives its nerve supply from both the vagus and the sympathetic.

The Thymus gland in cases of Status Lymphaticus being abnormally large may have a larger supply of nerves or may make a greater demand on those nerves it has or from its size may exert an undue pressure on its nerves. This excessive strain sets up an irritation of the nerve fibrils which render them more excitable and, thus, more responsive to any extra stimulus. The sympathetic branches communicate with those/



those going to the heart either in the cardiac plexus or in the cervical ganglia. The irritability of the one is conveyed to the other and this has its effect on the heart's action. Experimental work has shewn that stimulation of the sympathetic going to the heart has the effect of accelerating the heart. The acceleration is accompanied by increased force and so the action of the nerve is, in addition, augmentor.

The subject of an enlarged Thymus is, thus, weakened by the irritability of the sympathetic nerves to the heart, and any shock which is a sufficiently strong stimulus will affect chiefly the vulnerable part. We have a counterpart in the case of Asthma. Irritation of some distant organ may reflexly excite an attack. The bronchioles are the susceptible part and the whole storm is spent on them. So it is in cases of Thymus enlargement. The sympathetic nerves in the cardiac plexus are the susceptible point, and some stimulation which may pass unthought of, either peripheral or otherwise, may light up the attack, and the heart is the victim.

The rate becomes rapid due to the accelerator fibres in the sympathetic, and in the seven cases in which the pulse is recorded it has been so. In Case III it was uncountable, and in Cases IV, and V, it was almost imperceptible, while in Case VI it reached 156 per minute. In Cases I, II and VII, it ranged between 120 and 144.

But/



But, in addition to the accelerator fibres there are the augmentor fibres of the sympathetic, In Cases i, ii, and iii, there is special mention in the records of the discrepancy between the force of the heart and that of the pulse. The heart beat was disproportionately stronger than the pulse as felt at the radial artery. As these notes were not taken by the same observer, I think the value of them is greater.

The sudden stimulation of the sympathetic with the consequent rapidity of the heart causes shock, and this leads to a vaso-dilatation with a consequent lowering of blood pressure. In addition, the heart is stimulated by the augmentor fibres, but the blood-vessels are not and do not contract and, in consequence, the tension of the pulse feels low notwithstanding the forcible cardiac impulse.

I do not know how this discrepancy can be explained except by the stimulation of the augmentor fibres of the sympathetic. None of the other theories that have been put forward satisfactorily account for it. I have not seen mention of it in any other records, but this augmentation of the heart beat along with the acceleration, I consider, is an important point in diagnosis, and also in the elimination of the causation of the disease.

Overstimulation of the sympathetic leads to exhaustion, and, thus, we have in the later stages of the illness the heart beat becoming weaker in force. This/

This was noticed in Case I which was carefully watched during the course of the attack. On admission the beat was forcible and rapid while in the last few hours it became less strong, the right ventricle began to dilate, and there were all the signs of the failing of an overworked heart. In many of the post-mortem examinations there was evidence of dilatation of the right ventricle, and in Case I, leeches were placed over the praecordia to help in relieving the pressure.

The heart muscle showed cloudy swelling as is so general in all post-mortem findings but there were none of the advanced signs of a toxaemia.

It is probable ~~there~~<sup>at</sup> were the subjects of Status Lymphaticus examined when in apparent health a rapid pulse would be commonly found. This is to be explained on the hypothesis of a constant slight excitation of the sympathetic nerves to the heart owing to the pressure or irritation of them by the enlarged Thymus. The augmentation of the heart's action is to be looked for only in cases of excessive stimulus as is seen in the actual attack that results in Thymus death, but a rapid pulse in a child, the subject of the lymphatic diathesis, should be a suggestive and valuable diagnostic sign.

From the standpoint of Hyperthymisation the rapid pulse may be explained but not the discrepancy between the force of the pulse and the heart beat, and, again, if this theory were to hold true, it would be but right to expect minor attacks due to the excessive output of/  
of/



of secretion, but there is never a history of such.

The next point for consideration is the condition of the eyes. In Case I, which was under my own observation, the staring look amounting to exophthalmos, was a most noticeable feature. The pupils were widely dilated and did not react to light while the conjunctival reflex was sluggish, but in the later stages as the patient grew worse, the exophthalmos passed off, the pupils became less dilated, and the light reflex returned. How can these changes be accounted for? Do they not form in with the stimulation and subsequent exhaustion of the heart and can they not be attributed to the same cause?

In other two Cases (IV and VII) where the condition of the pupils is recorded there was dilatation. Unfortunately, the records have not been given in detail, and as the dilatation of the pupil may vary according to the stage the patient is in, one description of them does not give much help.

Purves Stewart in his "Diagnosis of the Nervous System" lays stress on the sympathetic innervation of the eye. He says "The Cervical part of the sympathetic chain has very special "oculo-pupillary" fibres. These supply the dilator pupillae, the non-striated part of the levator palpebrae superioris and the orbital muscle of Muller - a small bundle of non-striated muscle which lies behind the globe of the eye and bridges across the spleno-maxillary fossa at the/  
the/

the back of the orbit. The Cervical sympathetic also supplies secretory fibres to the submaxillary gland, and like the sympathetic elsewhere, it supplies fibres to the cutaneous blood-vessels, also (through the hypoglossal nerve) to the vessels of the tongue and lastly fibres to the sweat glands of the head and neck.

The pupil dilating fibres have a peculiar course which is important to remember. Arising from the pupillary centre in the medulla they descend in the lateral column of the spinal cord to the cilio-spinal centre in the lower cervical region. They emerge from the cord through the anterior roots of the first and second thoracic segments and enter the Inferior Cervical ganglion of the cervical sympathetic by white rami communicantes. They then ascend in the cervical sympathetic to the Gasserion ganglion and pass thence to the orbit (along the ophthalmic division of the fifth cranial nerve) and via the long ciliary nerve of the pupil. They do not traverse the ciliary ganglia".

It is, thus seen that the "oculo-pupillary" fibres enter the Lower Cervical ganglion and then pass up through the Cervical sympathetic. There is, accordingly, close interchange between these fibres and those going to the heart, and any irritation of the one will be communicated to the other. Stimulation of the sympathetic branches to the heart will excite a like stimulation/



stimulation of the cervical sympathetic generally. What are the effects on the eye? The muscle of Muller becomes firmly contracted and pushes the eyeball forward.. The involuntary fibres of the levator palpebrae superioris go into spasm and cause a widening of the palpebral fissure. The contractions of these two muscles produce a degree of exophthalmos and explain the protruding eyes and staring look which struck one so forcibly.

The marked dilatation of the pupil is also due to the stimulation of the sympathetic, and the absence of the light reflex can be explained on similar grounds. The tonic spasm of the dilator iris counteracts and prohibits any contraction set up by the stimulation of the third nerve and, thus, no reaction is seen.

Similarly with the conjunctival reflex. It was sluggish owing to the excessive contraction of the non striated portion of the levator palpebrae superioris which prevented the downward movement of the lid on applying the finger to the conjunctiva.

Prolonged stimulation of the sympathetic produced exhaustion and all the signs of excessive irritation passed off, and thus, we have the retraction of the protruding eyes, the contraction of the dilated pupils and the return of the light reflex.

The third point noticed in Case I was the profuse perspiration over the face and upper part of the chest as compared with the rest of the body. This, too, can/

can be explained by the stimulation of the cervical sympathetic as the sweat glands are supplied by the sympathetic and their secretion varies with the stimulation. Purves Stewart describes a case of paralysis of the sympathetic in a soldier who was wounded in the South African War. The bullet passed in front of the spinal column in the neck and afterwards there was no sweating on that side of the face, in addition to all the other signs of paralysis of the sympathetic. This is the exact opposite of stimulation where there is excessive perspiration.

The fourth point of discussion is the restlessness or fits. This is an almost constant sign and out of the 8 cases where the records were kept 6 were restless in varying degree, viz, Cases I, II, IV, V, VII, VIII, while 2 had fits Cases III and VI.

It is likely that the fits are an exaggerated condition of the restlessness and that both may be assigned to the same cause. The restlessness took the form not so much of the irritability seen in feverish conditions as of spasmodic contractions where the arms were flung about and became rigid and stiff at times. This condition is apparently due to some irritation of the cortex of the brain. It has been found experimentally that Cerebral Anaemia produces symptoms similar to these. Russmaul and Ternier tied the left subclavian and innominate arteries. The immediate symptoms were loss of consciousness and voluntary movements. These were followed in ten to forty-five seconds/



seconds by clonic spasm beginning in the muscles of the neck, then occurred dilatation of the pupils, respiratory gasps at longer and longer intervals and finally cessation of respiration. Orchansky says that the first effect of anaemia due to ligature of the carotid artery is to raise the excitability of the cord.

But how is this anaemia produced in Status Lymphaticus? Excessive stimulation of the cervical sympathetic causes spasm of the arterioles as they enter the brain and it is along these vessels that the sympathetic fibres chiefly enter the cranium. It has been shown that the sympathetic twigs do not accompany the vessels once they enter the brain substance, but they supply vaso-constrictor fibres immediately before their entrance and this governs the cerebral blood supply. Spasm of these arterioles leads to diminished supply of blood in the cerebral arteries and a consequent anaemia. The shock produced by the stimulation of the cervical sympathetic and the rapid action of the heart causes dilatation of the abdominal veins and this stagnation of blood aids in increasing the cerebral anaemia.

But as this overactivity of the sympathetic fails and exhaustion results, the spasm of the arterioles passes off and the cerebral anaemia gives way to hyperaemia. In mostly all the post-mortem examinations where a record of the brain is given there was marked congestion. Whether this latter hyperaemia produces any/

any symptoms it is difficult to say. Sir William Gowers says "Of all regions of cerebral pathology, that of congestion of the brain is the most obscure. We have little precise knowledge regarding it".

In Case i when the stage of exhaustion of the sympathetic was reached as seen by the dilating heart, the retraction of the protruding eyes, the contraction of the dilated pupils, the restlessness and movements entirely ceased. The cortical irritation was removed. The cerebral anaemia had passed off.

An interesting symptom, in that it was common to all except one, is vomiting. In Case i there was a history of bilious turns on slight provocation, such as even the shaking produced by a drive on a tramcar, but there was no vomiting during the fatal attack. Case V was the only one where there was no suggestion of it. In Case IV it was the chief source of complaint and in Case Viii after the tonsillotomy it was most distressing. It is peculiarly striking that it should occur so constantly, and one cannot help feeling that it is the condition of the heart that is to account for it. It may be explained on the same grounds as we explain vomiting in Diphtheria. It is the sign of a failing heart. The reflex irritation excites vomiting and in some it becomes violent.

From these clinical signs I came to the conclusion that the condition may be explained by irritation of the sympathetic nerves to the heart. It seems to embrace all the symptoms, and all the varied manifestations/



tions have been shown to be referable to it. It holds in points where the toxaemic theory breaks down.

Little or no mention is made in the literature of the subject of the different symptoms and signs that lead one to a diagnosis. From a study of the cases I have described, I think the following may be taken as the leading diagnostic ones.

- i Rapidity of the heart's action.
- ii Discrepancy between the force of the heart beat and that of the pulse.
- iii Protruding and staring eyes with pupillary changes.
- iv Restlessness which may culminate in fits.
- v Vomiting.

Should such symptoms show themselves, one should proceed to percuss out the Thymus to corroborate the suspicion of enlargement. It is, however, difficult in children to elicit a slight degree of dulness with any precision owing to the general resonance all over the chest. If the Thymus is distinctly enlarged, dulness should be detected in the majority of cases and its presence is always a great help in the making of a diagnosis.

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S U M M A R Y .



S U M M A R Y.

The Thesis is based on a study of nine cases in which Post-Mortem Examinations were carried out in all, and in three of which I made <sup>a</sup> microscopic examination of the different organs.

I have endeavoured to show that the four leading theories held regarding the causation of death in Status Lymphaticus fail to explain many of the symptoms as seen in these cases. These theories are

- i Laryngospasm
- ii Pressure on the Trachea
- iii Lymphatico-Chlorotic Constitution.
- iv Hyperthymisation.

The chief criticism I have brought against each may be briefly stated.

i Laryngospasm setting up a Thymus Asthma is not tenable in that none of the cases manifested throughout life any signs of Asthma. The absence of any such symptoms in a series of cases shown post-mortem to have definite thymus enlargement is sufficient in itself to discard this theory.

ii Pressure on the trachea, as the cause of death, does not get any support from these cases. The falling back of the head and the inability of the child to restore its position is held to lessen the antero-posterior diameter of the thorax and thus accentuate the pressure of the thymus on the trachea, but this additional factor can only apply to the very young, and in/

in only three out of nine cases was the age below nine months. There was no evidence in any of death by suffocation, as should be expected, if the theory of pressure were correct.

iii The "Lymphatico-Chlorotic" constitution was considered by Paltauf, who originated the term Status Lymphaticus, to be the predisposing cause. He held it lead to a condition of general weakness and any exciting cause was sufficient to produce death. If such were the case, we should expect to find on microscopic examination of the heart and other organs signs of fatty degeneration, but in the cases I examined microscopically there was no evidence of degeneration beyond early cloudy swelling which is seen in all post mortem examinations.

iv Hyperthymisation was the theory advanced by Escherich. He claimed that the excessive secretion of the thymus had a specially toxic action on the nervous system and any exciting stimulus was sufficient to set up such irritation as to cause death. Should this be the case, we should naturally look for minor attacks simulating the fatal one, but there is no such history in any of these cases.

Again, taking an analogy from Pneumonia, we find that when the toxaemia is severe the knee jerks are absent, while in only one out of five of our cases was it absent - this one was accountable for by other causes - and in some of the others it was even exaggerated. And, in addition, acetone which has been/



been shown to be an index of a toxaemia was absent in the case where it was tested for.

The microscopic sections I examined showed none of the disintegration of tissue seen in acute toxic conditions.

A study of these cases have led me to different conclusions, and I give the following short account of them.

I have shown that Dr Deard has demonstrated that the thymus gland is the parent source of the leucocytes and lymphoid elements in the body. I have applied this to the general hyperplasia of lymphoid tissue in Status Lymphaticus. The thymus has been overactive in its production of leucocytes and to meet this demand has hypertrophied while the lymphoid tissue has responded to the activity of the parent source and has enlarged correspondingly.

This, I consider, explains the hyperplasia of the lymphoid elements of the body.

As to the cause of death, I think the signs and symptoms during the fatal attack point to an irritation of the sympathetic nerves to the heart.

The attack may be noticed to pass through two phases due to (1) stimulation of the Cervical sympathetic and (2) to its exhaustion.

In the early stage, namely, that of irritation, the heart rate is exceedingly rapid.

This I have found to be a constant and invariable sign/

sign. It was present in all of the cases where the heart condition was described and in three, special mention was made of the disproportionate strength of the beat of the heart as compared with that of the pulse. This I take to be due to the excessive stimulation of the heart by the augmentor fibres of the sympathetic while the radial artery was relaxed due to the shock caused by such stimulation.

Then, there are the eye signs. In the earlier stages there are staring of the eyes amounting almost to exophthalmos, dilatation of the pupils, and diminution of the conjunctival and light reflexes. During this period there is marked restlessness which in an aggravated form may culminate in fits.

These manifestations, I have shown, may be explained by the excessive stimulation of the sympathetic to the heart being conveyed through the Cervical ganglia to the sympathetic branches to the eye and to the arteries to the brain.

Secondly, there comes the phase due to exhaustion of the sympathetic. The heart, although rapid, no longer beats forcibly, the protruding eyes retract, the pupils become less dilated and the restlessness ceases. There is vomiting which has been very constantly present and I think is due to the failing heart similar to what we find in Diphtheria. The patient becomes ashen grey or cyanosed and death ensues.

The thymus has pressed on the sympathetic or it has/



has made a greater demand on the branches which supply it through its unusual size, and this has probably set up the irritation which makes the nerve respond so readily to any exciting stimulus.

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